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Nitrogen Metabolism in the Brain of Rats
Subjected to the Action of Impulse Accelerations

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JPRS: 38,380

TT: 66-34806

28 October 1966

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- USSR -

FACILITY FOR 202

67-111111
(ACCESSION NUMBER)

7
(PAGES)

(NASA CR OR TMX OR AD NUMBER)

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NITROGEN METABOLISM IN THE BRAIN OF RATS SUBJECTED TO THE ACTION OF IMPULSE ACCELERATIONS

[Following is a translation of an article by Z. S. Gershenovich, A. Z. Gershenovich, L. A. Odnokrylaya, E. Z. Emirbekov and Ya. I. Veksler in the Russian-language journal Voprosy Meditsinskoy Khimii (Problems of Medical Chemistry), Vol 22, No 3, May-Jun 66, pp 262-264.]

Practical aviation and space travel involves the possible action of shock accelerations on human beings. The effect of these accelerations on human beings is being subjected to thorough investigation at present [1-3]. Research in this field is being conducted principally in connection with catapulting.

In various types of special work (geological prospecting, road construction work, etc.) the personnel must stay in structures providing protection from explosions. During the time involved, human beings may be subjected to impulse accelerations produced by the propagating explosion wave.

No information could be found in the literature concerning the effect of impulse accelerations on metabolic processes of the brain. Under the circumstances we determined in the work described the concentration of free ammonia, glutamine, glutamic acid, aspartic acid, and γ -aminobutyric acid as well as the amount of labile and firmly bound amido groups of the total proteins in the brain of animals subjected to the action of acceleration impulses. Determinations were carried out immediately after subjecting the animals to these impulses and three hours later.

The Method Applied

White laboratory rats weighing 130-160 g were placed into a special hermetically sealed chamber. An acceleration was

imparted to the chamber by the action of an explosion wave ($250-300 \text{ m/sec}^2$). The conditions with respect to the gas medium and the temperature of the environment to which the control and experimental animals were exposed during the period of preparation and during experiments were the same.

Within 15-20 min after the action of the acceleration, the rats were removed from the chamber and submerged into liquid air. A part of the animals were sacrificed three hours after exposure.

The brain without the cerebellum was isolated in a frozen state. After the membranes of the brain had been removed, the brain was triturated in liquid air and transferred in a powdered state for the precipitation of proteins to a test tube containing cooled 5% trichloroacetic acid. Precipitation was carried out within 20 min at a low temperature. The precipitate was separated by centrifuging. Ammonia according to Seligson [4], glutamine [5], glutamic, aspartic, and γ -butyric acid [6] were determined in the supernatant liquid.

The ammonia corresponding to labile and firmly bound amido groups of the proteins was determined in the precipitate after hydrolysis.

The total number of rats used was 90. The data obtained were subjected to statistical treatment.

The accelerations were arbitrarily subdivided into three ranges, weak (4-10 g), medium (11-24 g), and strong ($> 24 \text{ g}$).

Results of the Investigation

Upon action of an impulse acceleration of 4-10 g, there was a statistically reliable increase of the ammonia content in the brain of the animals (Table 1), while the concentration of glutamine (GL) decreased by 12% ($P < 0.002$). Notwithstanding the apparent deamidation of GL, the content of glutamic acid (GA) remained within the limits established for control animals. There was a tendency towards some increase in the content of aspartic acid, while the concentration of γ -aminobutyric acid (GAMA) remained unchanged.

Statistically unreliable data were obtained on changes in the content of labile and firmly bound amido groups of proteins. One may assume that the principal source of ammonia in this case was GL.

Three hours after the action of an impulse acceleration of 4-10 g, the concentration of ammonia dropped to values found

for control animals, while the concentration of GL also reached normal values and the content of GA decreased.

Table 1

Content of Components of Nitrogen Metabolism in the Brain of Rats (in mg%) Upon Action of an Impulse Acceleration of 4-10 g

(1) Исследуемые компоненты	(2) Контроль (24)	(3) Опыт	
		через 15-20 мин. (16)	через 3 часа (8-9) (5)
N аммиака (6)	0,86±0,01	1,68±0,05 $P<0,001$	0,84±0,07
Амидный N глутамина (7)	7,39±0,09	6,51±0,28 $P<0,001$	7,18±0,12
Глутаминовая кислота (8)	127±6,09	128±4,23 $P>0,5$	123±3,00 $P>0,5$
Аспарагиновая кислота (9)	36,4±2,42	39,6±2,3 $P>0,5$	40,8±2,38 $P>0,1$
γ-Аминomásляная кислота (10)	23,8±1,29	23,6±0,78	25,1±1,33 $P>0,5$
(11) N амидных групп суммарных белков (в мкмоль на 1 г сухого белка):			
лабильные (12)	125,1±3,3	127,6±3,0 $P<0,5$	121,2±1,59 $P<0,1$
прочно связанные (13)	286,4±11,3	280,6±8,8 $P<0,5$	278,2±8,4 $P<0,1$
сумма . . (14)	411,5±14,3	408,2±7,1 $P>0,1$	399,4±6,7 $P>0,1$

1 -- Components studied; 2 -- Control (24); 3 -- Experiment; 4 -- after 15-20 min (16); 5 -- after 3 hrs (8-9); 6 -- Ammonia N; 7 -- Amido-N of glutamine; 8 -- Glutamic acid; 9 -- Aspartic acid; 10 -- γ-aminobutyric acid; 11 - N of amido groups of total protein (in μ moles per g dry protein); 12 -- Labile; 13 -- Firmly bound; 14 -- Total.

Note: In all tables the figures in parentheses indicate the number of animals.

At an impulse acceleration of 10-24 g, the liberation of ammonia in the brain was activated to a still greater extent (234.5% with reference to controls: cf. Table 2). This amount was almost equivalent to the decrease in the concentration of GL. In connection with this, the amount of GA showed a statistically reliable increase by 8% and that of GAMA by 8.6%.

The amount of labile amido groups showed a statistically reliable decrease by 35.7%. The effect on firmly bound amido groups was more pronounced.

Table 2

Content of Components of Nitrogen Metabolism in the Brain
of Rats (in mg%) Upon Action of an Impulse Acceleration
of 11-24 g

(1) Исследуемые компоненты	(2) Контроль (24)	(3) Опыт	
		через 15-20 мин. (17) (4)	через 3 часа (9) (5)
(6) N аммиака	0,86±0,01	1,97±0,01 $P<0,001$	2,02±0,01 $P<0,001$
(7) Амидный N глутамина	7,39±0,09	5,57±0,06 $P<0,001$	5,40±0,07 $P<0,001$
(8) Глютаминовая кислота	127±0,09	137±4,50 $P<0,002$	118±7,40 $P<0,1$
(9) Аспарагиновая кислота	36,4±2,42	41,5±1,32 $P<0,1$	32,3±1,30 $P>0,1$
(10) γ-Аминomásлиная кислота	23,8±1,29	28,4±1,20 $P<0,05$	18,7±0,80 $P<0,01$
(11) N амидных групп суммарных белков (в мкмоль на 1 г сухого белка):			
(12) лабильные	125,1±3,3	80,4±1,28 $P<0,001$	77,2±2,30 $P<0,001$
(13) прочно связанные	286,4±11,3	282,2±9,1 $P>0,1$	267,4±8,4 $P<0,1$
(14) сумма	411,5±14,3	362,6±6,27 $P<0,01$	344,6±8,0 $P<0,01$

1 -- Components studied; 2 -- Control (24); 3 -- Experiment;
4 -- After 15-20 min (17); 5 -- After 3 hrs (9); 6 -- Ammonia
N; 7 -- Amido-N of glutamine; 8 -- Glutamic acid; 9 -- Aspar-
tic acid; 10 -- γ-aminobutyric acid; 11 -- N of amido groups
of total protein (in μ moles per g dry protein); 12 -- Labile;
13 -- Firmly bound; 14 -- Total.

Three hours after the action of impulse accelerations of medium strength, the amount of ammonia reached 242% as compared with the control. The concentration of GL dropped somewhat and the content of GAMA decreased by 34.2%.

As distinguished from the experiments described above, those of the 3rd series (at strong accelerations) were conducted in the summer.

Of the 10 rats used in the experiments with strong acceleration, three perished. The animals that remained alive were in a grave state. The content of ammonia in the brain increased markedly (Table 3), while the content of GL decreased considerably.

The content of GAMA increased greatly after the action of strong accelerations and amounted to 297% of the value for control animals.

Table 3

Content of Components of Nitrogen Metabolism in the Brain of Rats (in mg%) Upon Action of an Impulse Acceleration Greater Than 24 g

(1) Исследуемые компоненты	(2) Контроль (10)	Опыт (3)
		через 15-30 мин. (7) (4)
(5) N аммиака	$0,92 \pm 0,21$	$3,25 \pm 0,28$ $P < 0,001$
(6) Амидный N глутамина	$7,31 \pm 0,58$	$4,02 \pm 0,36$ $P < 0,001$
(7) Глутаминовая кислота	$145 \pm 9,43$	$132 \pm 9,03$ $P < 0,25$
(8) Аспарагиновая кислота	$37,0 \pm 4,45$	$31,9 \pm 1,22$ $P < 0,25$
(9) γ -Аминomásляная кислота	$20,0 \pm 1,64$	$59,4 \pm 3,17$ $P < 0,001$
(10) N амидных групп суммарных белков (в мкмоль на 1 г сухого белка):		
(11) лабильные	$144,4 \pm 8,01$	$80,7 \pm 2,01$ $P < 0,001$
(12) прочно связанные	$475,1 \pm 9,4$	$582,6 \pm 2,96$ $P < 0,05$
(13) сумма	$619,5 \pm 5,80$	$663,3 \pm 4,97$ $P < 0,1$

1 -- Components studied; 2 -- Control (10); 3 -- Experiment; 4 -- After 15-30 min (7); 5 -- Ammonia N; 6 -- Amido-N of glutamine; 7 -- Glutamic acid; 8 -- Aspartic acid; 9 -- γ -aminobutyric acid; 10 -- N of amido groups of total protein (in μ mole per g dry protein); 11 -- Labile; 12 -- Firmly bound; 13 -- Total.

In the resulting state of the organism, the total amount of amido groups contained in protein increased to a minor extent only. However, the ratio of labile to firmly bound amido groups changed considerably. In comparison with controls, the amount of labile amido groups decreased by 44.1%, while the amount of firmly bound amido groups increased by 22.5%.

Conclusions

1. Upon the action of impulse accelerations, changes took place in the content of ammonia and of some other compounds participating in the nitrogen metabolism of the rat brain.

2. As a result of the action of the accelerations, the concentration of ammonia in the brain increased considerably, while that of glutamine decreased. The content of glutamic acid remained at approximately the same level.

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Submitted
10 Sep 1964

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